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doi:10.1016/j.jtcvs.2009.04.034

Reply to the Editor:

We thank Dr Beghetti and colleagues for their thoughtful comments regarding the hemodynamic evaluation and further management of our recent case of atrial septal defect (ASD).[1] The management of patients with a degree of pulmonary vascular disease prior to shunt closure has been a matter of debate. In addition, recent studies demonstrating the efficacy of oral vasodilators in pulmonary vascular disease associated with congenital systemic-to-pulmonary shunts[2,3] have fueled an uncertainty of vasodilator pretreatments prior to shunt closure.

Despite optimized medical treatment with diuretics, antibiotics, and oral anticoagulant over 4 months since the first medical contact, our patient was severely dyspneic, with elevated atrial pressures, a pro–brain natriuretic peptide serum level of above 4000 pg/mL, and a dramatically limited 6-minute walking distance (6-MWD) of 150 m. In fact, based on the hemodynamic assessment alone, the patient was admitted to the surgical ward for ASD closure. However, surgeons refused the operation based on the patient's overall clinical profile and frailty. A 10-month treatment with bosentan on top of supportive treatment with diuretics and anticoagulation effectively decreased shunt flow and lowered pulmonary vascular resistance by 140 dynes $\cdot$ s^{-1} $\cdot$ cm^{-5} and markedly decreased atrial pressures, biomarkers, and 6-MWD in the presence of a mild arterial desaturation.

We do agree with the discussants that taking into account left atrial pressures, pulmonary arteriolar resistance was about 3 Woods. In addition, the pulmonary-to-systemic resistance ratio under oxygen and nitric oxide was <0.33 (in the patient, this ratio was 0.11), a threshold pediatric cardiologists have labeled as a criterion conveying a good prognosis after closure of the shunt.[4] Still, data in adult patients with congenital heart disease are lacking, and the criteria of a complete hemodynamic responder status in adults were not fulfilled in this case.[5] Because hemodynamic testing is a routine procedure in adult pulmonary vascular centers, we do rely on these data in the absence of firm evidence indicating their uselessness in adults with congenital heart disease. Moreover, children are usually examined under general sedation/anesthesia. For these and other reasons, it is evident that the hemodynamic response pattern in children is different from that in adults[6] and that hemodynamic criteria in children may not apply to elderly adults. Furthermore, later assessments after surgery in the patient under discussion illustrated a degree of persistent pulmonary vascular disease with a pulmonary arteriolar resistance of 530 dynes $\cdot$ s^{-1} $\cdot$ cm^{-5}, despite active treatment with bosentan.[1]

The main value of this report is to provoke discussion, because due to its single case nature, surgery in the absence of bosentan cannot be repeated.

We submit that our invasive procedure was based on an integrative clinical and hemodynamic approach and guided by numbers, rather than the reverse. Controlled data to guide a ''targeted treatment-and-repair'' strategy in adult patients with congenital heart disease are needed.

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QUALITY OF LIFE IN PATIENTS WITH PROSTHESIS–PATIENT MISMATCH

To the Editor:

I read with interest the article by Moon and colleagues[1] and thank the authors for their contribution to the continued debate on the issue of prosthesis–patient mismatch (PPM).

PPM is an important topic in current cardiac surgery, and there are several discrepancies and contrasting publications about the effect of PPM on postoperative outcome.[2,3] Some of these controversies are discussed in the accompanying counterpoint article by Dr Feindel.[4] In their work Moon and colleagues[1] provide important insight into the management of aortic valve replacement (AVR) with bioprostheses in patients older than 70